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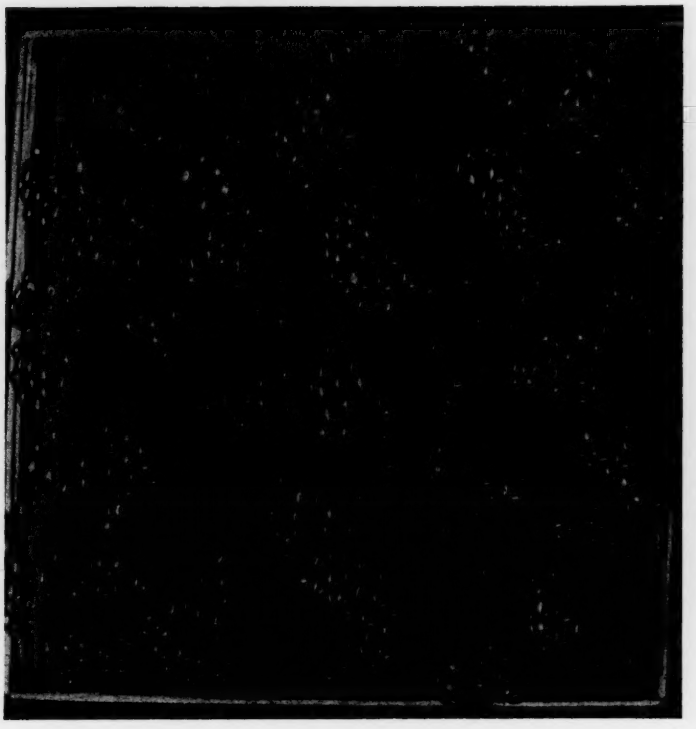

KEY WITNESS IN THE MAMMOTH MYSTERY
(SEE PAGE 105)

Growth Hormones and the Meristem

A Proposal to Revise Blood Group Nomenclature

Will Elizabeth's Heir Have Hemophilia?

Swine Coat Color

VOLUME 39 APRIL, 1948 NUMBER 4

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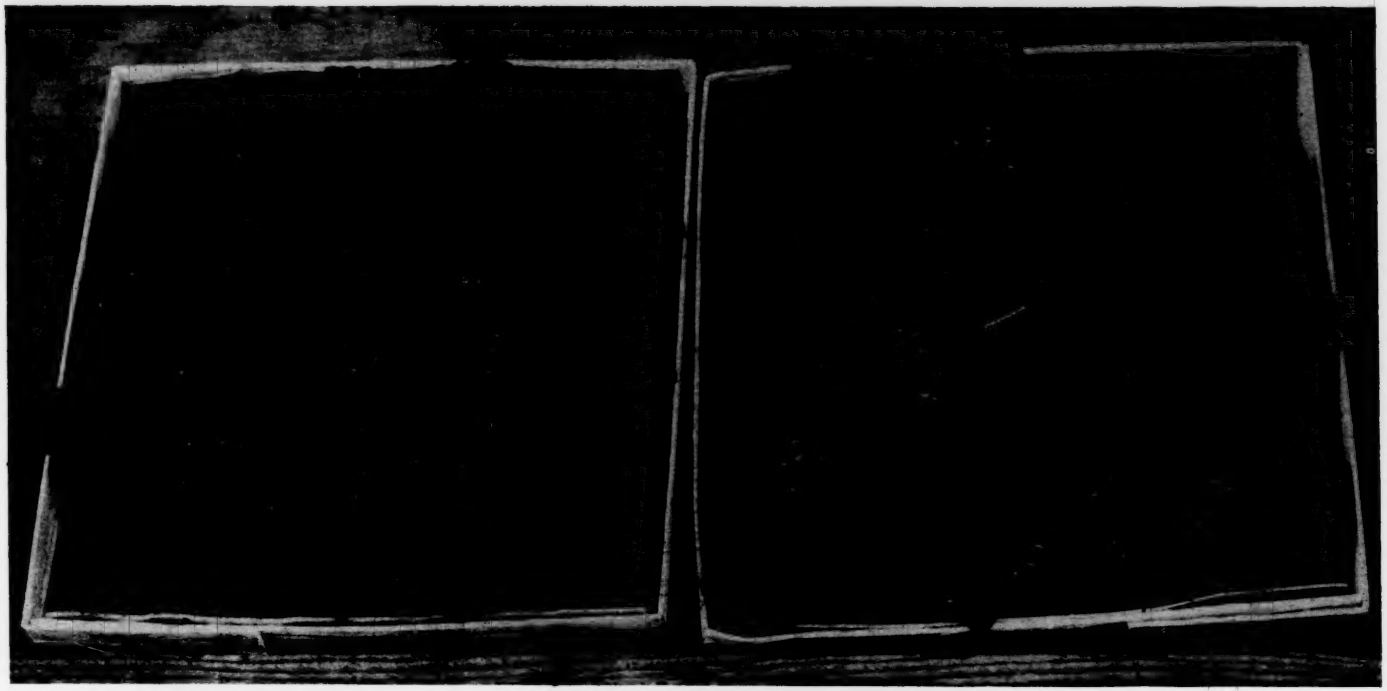


LOGAN AND PHENOMENAL

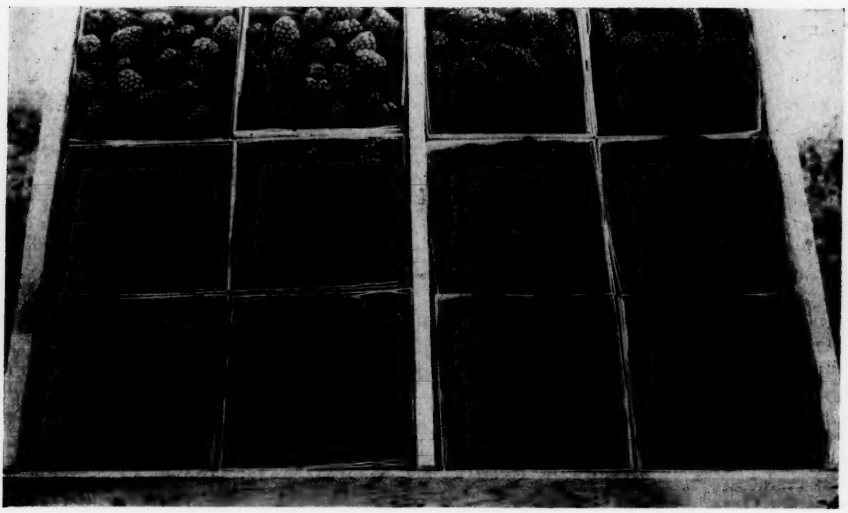
Figure 1

Pint boxes of Logan and Phenomenal berries approximately natural size. The Logan and Phenomenal blackberries are two varieties originating in California whose origin has been something of a mystery. Judge J. H. Logan introduced his variety in 1881 and Luther Burbank followed shortly thereafter with the Phenomenal, like the Logan but slightly larger and subject to the "dwarf disease." Cytological and genetic evidence indicates that Judge Logan's account of the parentage of this berry is correct though it has been questioned for many years. It is a hybrid between a California wild blackberry and a European variety of red raspberry with two sets of raspberry chromosomes.

ORIGIN OF THE LOGAN AND THE MAMMOTH BLACKBERRIES

New Genetic Evidence Regarding This Perennial Horticultural Mystery

GEORGE F. WALDO AND GEORGE M. DARROW*



THE LOGANBERRY AND THE MAMMOTH BLACKBERRY

Figure 2

The pint boxes are four inches square so that the individual Mammoth berries average fully an inch in length. The application of new cytogenetic techniques to blackberry breeding has definitely established the origin of the giant forms and opens the way for the controlled production of superior new varieties.

THE Loganberry, or Logan blackberry, has long been an important fruit in the Pacific Coast States. Its distinctive flavor and the high flavors of its related varieties, Young, Boysen, and Cascade, and their adaptation to canning, freezing, preserving, and juicing have given this group considerable importance. The Logan has also become a commercial variety in England, in France, and in Australia. The cash value of the fruit of this variety produced since its origin in 1881 may reach a total of $50,000,000. The chief limitations of the Loganberry are its very high acidity, making it too acid to be

*Associate pomologist and principal pomologist, respectively, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture. Published with the approval of the Director of the Oregon Agricultural Experiment Station as Technical Paper No. 518. Contribution of the Department of Horticulture. The writers are grateful to George M. Roberts, foreman, and the Oregon Agricultural Experiment Station for the care and facilities in growing the plants in this investigation.

eaten as a fresh fruit unless dead ripe, its susceptibility to the leaf spot fungus, and its lack of hardiness. Hence its culture in the United States has been limited to the milder parts of the Pacific Coast, and it has been chiefly valuable for canning and preserving.

An understanding of the origin of the Logan would be useful in breeding programs to increase the number of varieties with the high flavors of this group. There was no doubt in the mind of the originator of the variety as to when and where he produced it. Sixty-seven years ago (1881) Judge J. H. Logan planted seed of the Aughinbaugh, a pistillate selection of the native western blackberry, in his garden in Santa Cruz, California. From this mass culture grew a red-fruited seedling which was called the Loganberry, or Logan blackberry. Logan also introduced others of his seedlings, one of which was called the Mammoth, which has the longest fruit of any variety ever grown.[9] Other varieties that may possibly have come from his, or Burbank's, work are the Boysen,* the Black Logan, and the Kosmo. Burbank followed Judge Logan by introducing the Phenomenal, similar to the Logan though slightly larger. It produces many double berries and is subject to the "dwarf" disease.

Judge Logan's version of the origin of the Logan was questioned by breeders because the Logan can be reproduced from seed, like a true species. Some of its seedlings ripen earlier, some later; some produce sweeter fruit, some smaller, some larger; but they all closely resemble the Logan.[3] The parentage of the Logan as given by Judge Logan himself and repeated recently by his wife, Mary E. Logan,[10] is this: "The Loganberry is a cross between the California wild [Aughinbaugh] blackberry and the old Red Antwerp, or a European variety of raspberry resembling the Antwerp. The Mammoth blackberry is a cross between the California wild blackberry and the Texas Early (Crandall) blackberry." Studies by Ness,[7] by Darrow and Longley,[3] and by Brown[1] added information to explain the Logan's peculiar behavior, but furnished no conclusive proof as to how it originated. Crane[2] and Thomas[11] maintained that the Logan, a hexaploid, originated as the result of the pollination of an octoploid blackberry with an unreduced (diploid) red raspberry pollen grain (possibly of Red Antwerp). They reported growing ten seedlings of an octoploid Pacific Coast blackberry crossed with the autotetraploid Hailsham red raspberry. The seedlings were fertile and were reported to resemble the Logan in essential botanical characters: they were also hexaploid, and 45 seedlings from them also were hexaploid and fertile. Thomas[11] suggested that some of the chromosome sets from the native western blackberry are closely related to those of the raspberry and pair freely with them. In contrast to the fertility of the octoploid × tetraploid seedlings, the hybrids of octoploid blackberry × the diploid Superlative red raspberry were highly sterile.

At the Oregon State Experiment Station, Corvallis, Oregon, collections of the wild trailing blackberries of that region had been made for many years and used in crossing with Logan, Boysen, and eastern blackberries. Among these were some plants known to be octoploids and hexaploids, and others of unknown chromosome number from the coast of northern California. In 1944 this group of plants was crossed with the La France, a tetraploid red raspberry of the European strain, similar to the Hailsham used by Crane.[2] One selection, Jenner #1, was also crossed with the Eldorado, a tetraploid erect blackberry. The resulting seedlings came into full fruiting in 1947. Seedlings of the first cross were similar to the Logan, which indicates that the Logan is actually a hybrid of

*According to H. M. Butterfield (private letter) the Boysen is not distinguishable from the Lubbenberry, a berry that traces back to San Leandro, California, in the 1920's. It resembles the "Humboldt" listed by Burbank in his catalogue for 1894 and said by him to be a cross of the native trailing blackberry and the Cuthbert raspberry and coming from the same lot of seed as the Phenomenal.

THREE PROMISING SEEDLINGS

Figure 3

A—Fruit cluster of Branstetter × La France. This represents a cross between a western octoploid blackberry and a European raspberry. The fruit resembles the Logan in appearance and quality. *B*—A seedling plant, fruiting heavily, of the cross Jenner-2 × La France. *C*—A seedling plant of the cross Jenner-1 × Eldorado. Both of these plants have qualities that encourage us to expect fine results from such crosses.

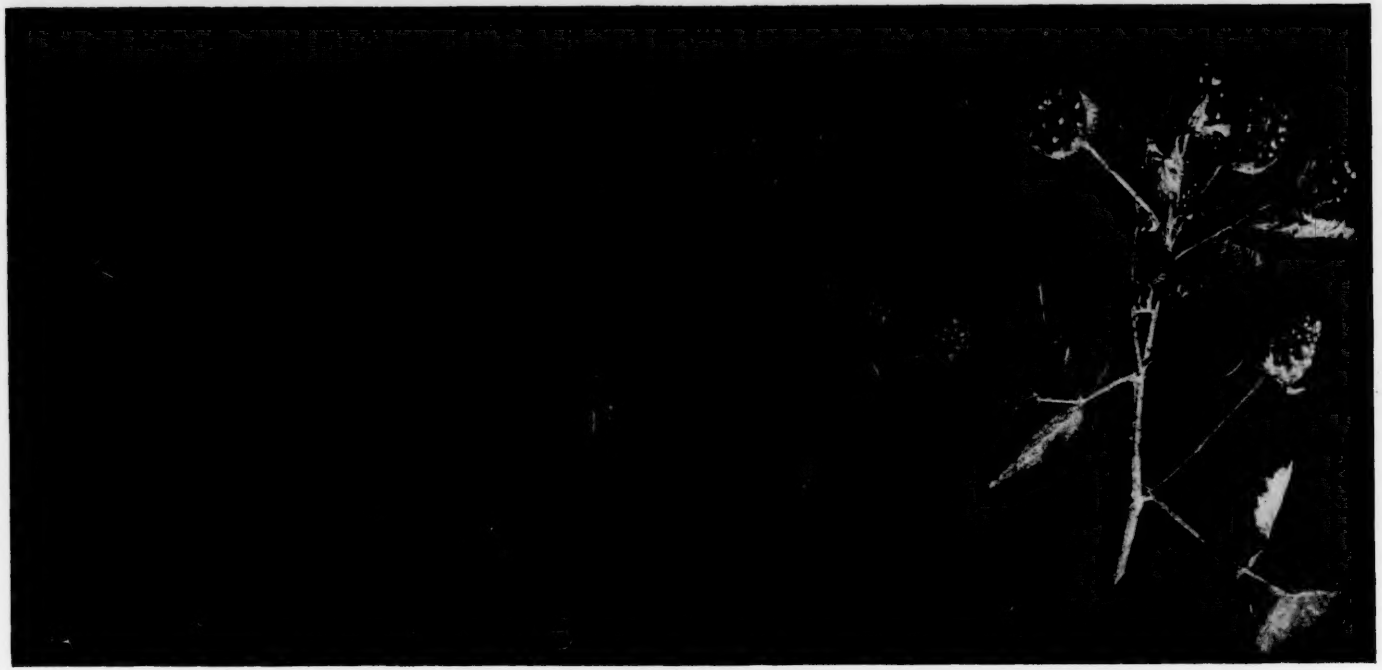
the Pacific coast native trailing blackberry and a red raspberry, as was maintained by Crane and Thomas. Seedlings of the native blackberry × Eldorado blackberry indicate that the Mammoth is in all probability an Aughinbaugh × Crandall blackberry hybrid.

Hybrids of Blackberry × La France Red Raspberry

Table I, at right, lists the crosses and seedlings growing in the field in 1947, according to their vigor. In addition to those listed as weak or dwarf, many more were discarded as lacking stamina or size at the time of planting, and still others died after planting.

TABLE I. Seedlings of crosses between wild trailing blackberries and La France red raspberries classified as to vigor. *A* = very vigorous; *B* = average vigor; *C* = weak; *D* = very weak or dwarf (per cent).

Crosses	Number set	Plants maturing	Seedlings classed as to vigor A	B	C	D
Jenner-1 × La France	78	66	13	41	35	11
Jenner-2 × La France	115	98	12	36	23	29
Branstetter × La France	82	79	33	47	19	1
Agate Beach (8x) × La France	52	45	9	53	16	22
Point Arena (8x) × La France	77	64	6	52	26	16
Patrick Point × La France	34	33	9	36	36	19
Ideal Wild (8x) × La France	59	44	5	45	45	5
	497	429				



FRUIT CLUSTERS OF LOGAN AND BLACKBERRY RELATIVES

Figure 4

To the left is a cluster of the Logan at an early stage of development. In the center is a cluster of the Snyder, an eastern blackberry of the type which is useful in producing polyploid crosses. To the right is a cluster of the Ideal Wild (Santiam) blackberry, an octoploid selection of the western trailing blackberry. The Logan is derived from a cross of a western blackberry of the Ideal Wild type with the European red raspberry. Controlled crossing of such raspberries and their artificial polyploidization offers great promise for the production of superior new varieties.

The wild trailing blackberry selections used as parents all came from points along the northern California coast with the exception of Ideal Wild, a selection of the native wild trailing blackberry made near Salem, Oregon. The chromosome number of the Ideal Wild and Point Arena selections has been reported as octoploid. Though Agate Beach was reported as hexaploid[6], the plants used in this work were octoploid. Seedlings of Branstetter × La France were outstanding for their vigor. The wild blackberry Branstetter came from the Branstetter ranch just north of Cape Mendocino, California.

Sex Inheritance

Table II lists the percentage of pistillate and of perfect-flowered plants together with the number that did not bloom.

This table shows that in general the cross of the California pistillate blackberries × the La France raspberry produced pistillate and perfect-flowered plants in about equal numbers. Thus, hexaploids resulting from four genoms from the native blackberry plus two genoms from the red raspberry give a fair approximation to a 1:1 sex ratio. This is the same as that obtained in crosses of pistillate with perfect-flowered strawberries where both parents are octoploids. The seedlings of the perfect-flowered Oregon selection Ideal Wild × La France were all perfect-flowered. None of the plants that produced flowers was completely sterile so that there were no pure males, as in the wild Pacific Coast blackberry.

TABLE II. Sex of wild trailing blackberry × La France red raspberry seedlings.

Crosses	Blooming		Non-blooming
	Pistillate Percent	Perfect Percent	Number
Pistillate × La France:			
Jenner-1 × La France	48	52	23
Jenner-2 × La France	44	46	10
Branstetter × La France	55	45	3
Agate Beach (8x) × La France	60	40	2
Point Arena (8x) × La France	49	51	10
Patrick Point × La France	41	59	5
Average	49	51	
Perfect × La France:			
Ideal Wild (8x) × La France	0	100	5

Fruit Color Inheritance

The fruit color of all seedlings was deep red, close to that of the Logan and having about the same range as that of Logan seedlings. Berries of the Branstetter × La France cross were lighter red than those of the other crosses (Figure 3*A*). None was black-fruited. Apparently fruit color represents a blending of the color of the two parents.

Separation of Fruit

In general there was a range of separation of the fruit from the stem from that of the blackberry to that of the raspberry (see Table III). Few fruits separated as easily as most raspberries do, but when fully ripe some came off the receptacle as easily as does the La France. Many were difficult to pick in any way. In many cases part of the receptacle was left on the plant and the remainder in the berry. The receptacle or core of the fruit of many seedlings was relatively hard.

The greatest percentage of seedlings with fruit separating easily from the stem, was found in the Bransetter × La France cross. Those crosses giving the largest proportion of seedlings with fruit-separation like the raspberry were with Jenner-1, Jenner-2, and Ideal Wild. The Nessberry,[4] a raspberry × blackberry hybrid from Texas, with two genoms from each of its raspberry and blackberry parents, also separates with difficulty.[7] The Logan and nearly all its seedlings,[3] separate like a blackberry, none like the red raspberry. B. M. Young, originator of the Young dewberry, has suggested (personal letter) that ease of separation of the parents

TABLE III. Ease of separation of fruit in seedlings of wild trailing blackberry × La France red raspberry.

Crosses	Easily from stem as in Loganberry Percent	Difficult or intermediate Percent	Separation more like raspberry Percent
Jenner-1 × La France	10	18	72
Jenner-2 × La France	17	43	40
Branstetter × La France	76	22	2
Agate Beach (8x) × La France	46	46	8
Point Arena (8x) × La France	48	52	0
Patrick Point × La France	50	46	4
Ideal Wild (8x) × La France	12	38	50

might largely determine the mode of separation of the seedlings, and this seems to hold true. He made many raspberry × dewberry crosses.

Fruit Size

Only an approximate record of berry size (by weight) of seedlings of the different crosses was possible, because of the small amount of fruit and the difficulty of picking it. There was, however, considerable difference in the size of berries on different seedlings. The averages given in Table IV are based on only a small number of plants.

Although the Branstetter fruit averaged only 1.6 grams per berry, the seedlings of the Branstetter × La France hybrids averaged 4.8 grams per berry, with one seedling averaging as much as 6.2 grams per berry. This latter weight is greater than the average of these berries and was greater than that of the Logan but less than that of the Boysen. The seedlings of the Jenner-1 and Jenner-2, hybrids with La France, also bore much larger fruit than did either female parent. A heavily fruiting plant is shown in Figure 3*B*.

Flavor

In general the flavors of the seedlings suggested those of the Logan and of the native wild blackberry. Some were very tart; others were very mild, lacking acid. Some had high aroma; others very little. None had unusual or new flavors different from those of the native blackberry, the Logan, the Boysen, or the Young, or crosses of these formerly grown; and none had as fine flavor as the Cascade.

TABLE IV. Average size of berries of seedling progenies and parents as measured in grams per berry.

Cross or variety	Number of seedlings	Average weight	Weight range (gm)
Jenner-1 × La France	5	4.2	3.6 to 5.0
Jenner-2 × La France	3	3.2	2.5 to 3.8
Branstetter × La France	12	4.8	3.4 to 6.2
Point Arena × La France	6	3.9	3.1 to 5.3
Patrick Point × La France	5	3.6	3.1 to 4.7
Jenner-1		2.0	
Jenner-2		2.2	
Branstetter		1.6	
Boysen		7.0	
Logan		5.7	

Seeds

Seediness was determined by weighing a sample of berries (usually 20) and the cleaned seeds derived from them. The ratio of these weights (expressed as a percentage) measured the "seediness" of the samples. Seed size was determined by weighing 100 seeds from each plant to obtain the average weight. Table V gives the values thus obtained.

The wild blackberry parents in general had medium to small seeds, though not so small as those previously reported for the wild blackberry.[4] However, the percentage of seed per berry was relatively high in plants from California. The Ideal Wild from Oregon was notable for both a low seed percentage and small seeds; its seedlings had very small seeds.

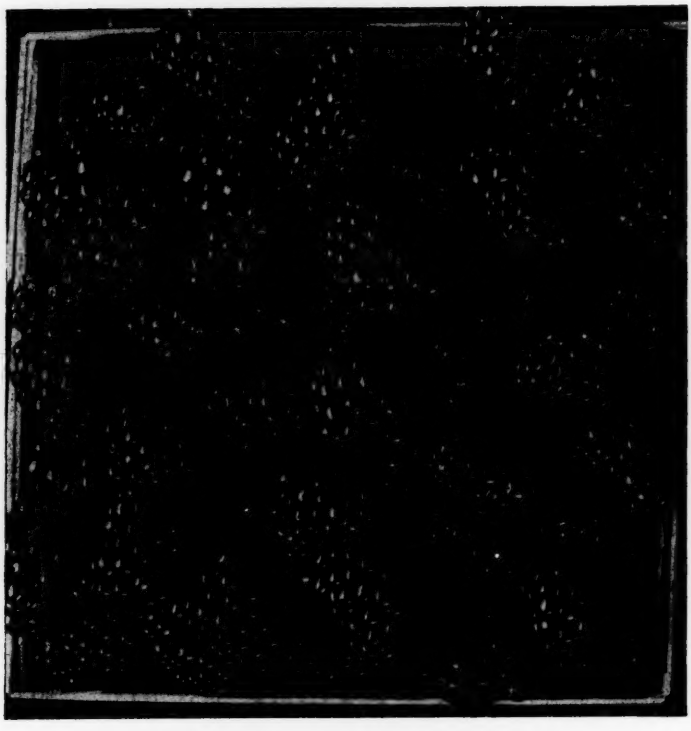
In general, the seedlings examined had a seed size close to that of the Logan and Logan seedlings,[4] and much smaller than that of the Boysen. There was considerable difference in seediness in the seedlings of the three crosses measured. Branstetter × La France seedlings were the most seedy, and Patrick Point × La France seedlings were the least. Other factors affecting seediness, such as softness of seed and attachment of flesh, were not studied.

Glandular Hairs and Prickles

Ten seedlings from each cross were examined for glandular hairs on the calyx. No hairs were found, however, except in the crosses with Jenner-1 and Jenner-2, where they ranged from none

TABLE V. Seediness and seed size in blackberry hybrids and varieties.

Cross or variety	Number of seedlings	Average seediness	Avg. weight single seed (mg.)
Branstetter × La France	12	4.26	2.78
Jenner-1 × La France	11	3.94	2.80
Patrick Point × La France	10	2.95	2.11
Agate Beach (8x) × La France	10	.....	2.66
Jenner-2× La France	10	.....	3.16
Point Arena (8x) × La France	10	.....	1.99
Ideal Wild (8x) × La France	11	.....	1.96
Branstetter		6.28	2.10
Jenner-1		5.07	2.70
Jenner-2		4.98	2.30
Boysen		4.67	4.70
Logan		3.90	2.50
Ideal Wild		2.39	1.80



THE ELDORADO BLACKBERRY

Figure 5

The Eldorado is a tetraploid blackberry variety which is important in the eastern United States. It has been useful as a parent in hybrids to produce superior polyploid varieties.

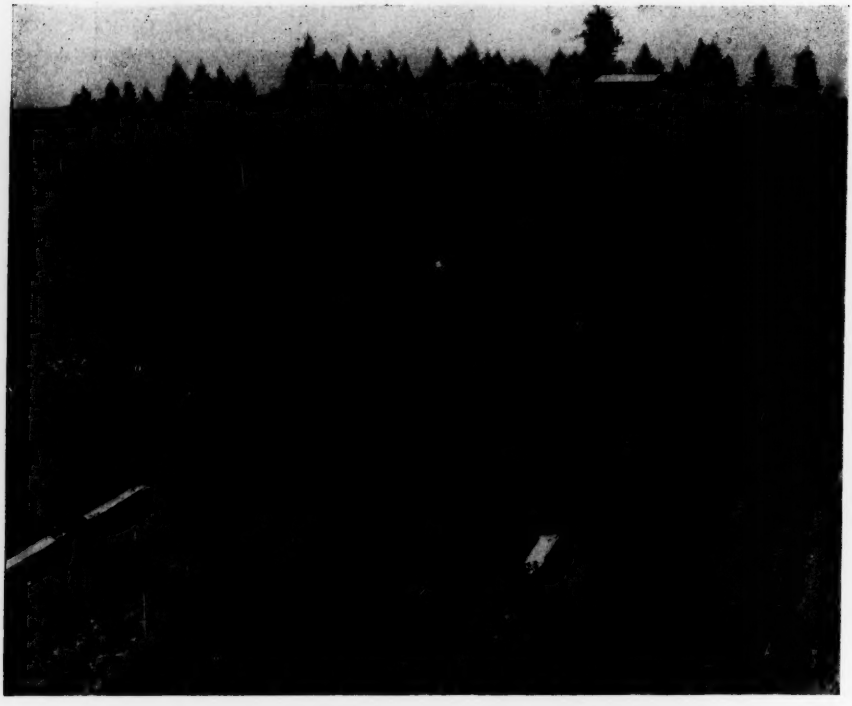
to many. Results of an examination of the parent blackberry varieties and of the Logan for glandular hairs were as follows:

Agate Beach	— none
Branstetter	— none
Jenner-1	— very many, dense
Jenner-2	— very many
Point Arena	— very few
Patrick Point	— very few
Logan	— none

The prickles of seedlings of three crosses were also compared with those of the Logan. The Branstetter × La France hybrids had notably softer prickles but the number and length of prickles of almost all seedlings were very much like those of Logan.

Hybrids of Jenner-1 Blackberry × Eldorado Blackberry

Jenner-1 was the only octoploid blackberry crossed with the tetraploid Eldorado. Of this cross, 223 seedlings were set and 171 grew to maturity. There were 71 pistillate, 76 hermaphrodite, and the remainder failed to bloom. About half the plants were vigorous, and a quarter were very weak or dwarf. About a tenth of the surviving seedlings had vigor equaling that of the Mammoth (Figure 3 *C*), and resemblance to this variety was quite marked in both leaf and cane characteristics, and even more striking in the prickles of most of the seedlings.



A FIELD OF LOGAN BLACKBERRIES IN OREGON

Figure 6

Since its introduction nearly seventy years ago, the Logan berry has enjoyed a steady popularity. Total cash value of this fruit has been estimated to amount to nearly fifty million dollars. It is grown in the northwestern states, in England and Australia.

The seedlings of this cross showed notable variation in the season of ripening. Many began ripening much earlier than the Logan or the Mammoth, and many ripened later. Only one, however, ripened as late as Eldorado. The berry size of the seedlings averaged smaller than that of Mammoth or Logan, though some were about equal in size. The berries of all seedlings were a bright black color and very attractive (Figure 3*C*). All picked off easily, like blackberries. The shape was variable, many being long like the Mammoth. Almost all had good flavor; some had a flavor very much like Eldorado and some a delightful combination of the high flavor of the native blackberry with that of the Eldorado.

Berries from 15 plants were studied for seediness. Seediness ranged from 3.13 to 5.27 percent, averaging 3.77 percent. The percentages for Logan and Mammoth were 3.90 and 3.26, respectively, while that reported[4] for Eldorado (Stuart) was 3.84. The seed weight was relatively low, ranging from 1.30 to 2.70 mg. per 100 seed and averaging 1.97 mg. The weight of seed of Logan and of Mammoth was 2.50 mg., and that of Eldorado was 2.55 mg.

Discussion

Seedlings from crosses of octoploid native western trailing blackberries with

the tetraploid, relatively tender European red raspberry La France show a close resemblance to the Logan, while the seedlings of a western trailing blackberry from Jenner, California, crossed with the tetraploid Eldorado resemble the Mammoth blackberry.

The characteristics of the seedlings obtained in these crosses indicate that true crosses were obtained. In the crosses where the La France red raspberry was used, the red raspberry parentage is indicated by the color and flavor of the fruit as well as by the difficulty and mode of separation of the fruit from the plant. No seedlings having either distinctly red raspberry or western native blackberry type of plant or fruit were obtained. Likewise, in the Jenner-1 × Eldorado seedlings the Eldorado parentage showed in fruit flavor, firmness, and size and sometimes in cane and leaf characteristics.

It is concluded from these observations that the Logan and Mammoth did originate from the parents suggested by Judge J. H. Logan and later by Crane and Thomas. Judge Logan was extremely fortunate in obtaining his one red-fruited seedling. Under normal conditions, diploid pollen is rarely produced by ordinary red raspberries. Either some special condition caused the formation of great numbers of diploid pollen grains, or by chance one of the rarely formed unreduced pollen grains reached the pistil of an Aughinbaugh flower in Judge Logan's garden. Furthermore, the resulting seedling had a combination of productiveness, large fruit size, ease of picking, and vigor of plant that was not found among the nearly 500 similar seedlings produced by the crosses here reported.

To greatly improve on the Logan as a horticultural variety, it may be that making autotetraploids of such varieties as Washington, Latham, or Willamette and crossing them with octoploid blackberries might give much greater hardiness.[12] Crosses of autotetraploids of the Sunrise red raspberry with the octoploids might give greater disease resistance.

Einset[5] found that two tetraploid eastern species of blackberries at meiosis behaved like autotetraploids. The present cross of Jenner-1 × Eldorado gave a progeny many of which were superior in several characteristics to the Mammoth. While the Aughinbaugh was probably much superior to the selections of the native trailing blackberry used, the Crandall (Texas) was probably a less desirable pollen parent than Eldorado. This suggests that the crossing of other tetraploid varieties of blackberries, such as Lawton and Mersereau, with octoploids, might yield valuable new combinations.

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BLOOD GROUP NOMENCLATURE

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NO uniformity and no conformity to established rules exists relative to the assignment of names or symbols to the genes which have been shown to be directly responsible for specific human variations. This is unfortunate. The lack of uniformity and of conformity has led to confusion and to the impression on the part of many people that little or no exact human genetic knowledge is at hand, when actually much precise information is available.

An international committee on human genetic nomenclature should be established which would agree on names and symbols for human phenotypes and genes. Adherence to its decisions should then be encouraged or demanded. There is some hope that such a committee may be formed sometime in the near future, but so far very little progress has been made towards its establishment. In the meantime the best that can be done is for individual geneticists to suggest and encourage the adoption of names and symbols which are descriptive, practical, and in conformity with certain rules which geneticists have found desirable to follow in their naming of genes which are responsible for genetic variation among lower forms.

It is not my intention in the present paper to outline all the rules which should be followed, or to suggest names and symbols for all of the human phenotypes and genes which are recognized. I shall concern myself only with the phenotype and gene nomenclature of the three sets of blood variations which are frequently discussed in laboratories and in the literature and whose nomenclature is in the worst state of confusion.

The A-B Blood Groups

The A-B blood groups were discovered in 1900-'01. They were given Roman numeral names by Jansky and by Moss. Unfortunately, the No. I of Jansky corresponded to the No. IV of Moss, and the No. IV of Jansky to the No. I of Moss. Both systems were used for a time. This led to considerable confusion. A committee of the League of Nations finally agreed upon a letter system, which is now known as the International A-B Nomenclature. According to this system the four major A-B blood groups are known as AB, A, B, and O. Fortunately this system is now used almost universally. A few clinicians continue to refer to the groups by number. This practice should be frowned upon.

That the A-B blood groups have a hereditary basis was first suggested by Epstein and Ottenberg in 1908. Two years later von Dungern and Hirszfeld postulated two pairs of autosomal alleles, with a dominance relationship within each pair. In 1925 Bernstein pointed out that three autosomal alleles with two of them dominant over the third but neither of the first two dominant over each other, would also account for the four phenotypes. His hypothesis has been tested extensively and is now almost universally accepted as the correct one.

Bernstein did not suggest appropriate names for the three alleles which he postulated. Some investigators have called them *A, B* and *O*. This is an unacceptable system of allelic names. Geneticists have found it extremely desirable to have a particular gene locus referred to by a single letter (or set of letters). In accordance with a partial list of the recognized rules, some geneticists have used the symbols *A*, A^B, and *a*. This is not a completely unacceptable system, but there exists the objection that the locus symbol *A* is identical with the name of one of the four phenotypes and emphasizes this phenotype, which does not deserve greater emphasis than does at least one of the other three. Fur-

thermore, the allele symbol A^B suggests the phenotype AB which, of course, it should not. The two may even be mistaken.

In the classroom and in several published papers I have used the symbols I^A, I^B, and i. I chose the letter I (capital or lower case) as an abbreviation of the word isoagglutinogen. Only one objection has been raised to this system, namely, that the capital letter I suggests the No. I of the old and almost universally discarded Roman numeral system of names for the A-B phenotypes. This is not a very strong argument, but I am willing to discard my proposed system in favor of another one, which I believe may have a chance of becoming universally accepted. It is the use of the letter L (capital or lower case) as the locus symbol. The reason why I believe that this symbol may become or should become universally accepted is that it commemorates the name of Dr. Landsteiner. What could be more appropriate? According to this proposal the genotypes of the four major A-B blood groups would be represented as follows:

Blood Group or Phenotype	Genotype
AB	L^AL^B
A	L^AL^A or L^Al
B	L^BL^B or L^Bl
O	ll

The symbols of genes responsible for subgroups of A or B can readily be developed by adding numbers to the superscripts (Ex. L^{A1} etc.). Two objections could be raised to the use of the letter L or l as the locus symbol but neither seems to be a serious one. One possible objection is that the letter l is often used for the symbol of a lethal gene and should be reserved for this type of gene. The other is that the typewritten letter l is identical with the typewritten number 1.

The M-N blood types were discovered by Landsteiner and Levine in 1928. These two investigators also demonstrated that these three blood types are inherited and that they involve a pair of autosomal alleles with no dominance. Landsteiner and Levine did not suggest appropriate names for the two alleles and have not insisted upon any particular set. Some investigators have called them M and N. This is an unacceptable usage for the reasons outlined earlier, namely, that a locus should not be represented by two different letters. Others have used the symbols M and m. Several objections may be raised against this system. One is that the locus symbol M gives undue emphasis to one of the two coordinate antigens, neither of which is known to be the original one. Furthermore, the use of capital and lower case letters suggests dominance which does not exist.

For several years I have used the symbols A^M and A^N; the letter A was chosen as an abbreviation of the word agglutinogen. Objections have been raised to this locus symbol on the ground that it suggests blood group A of the A-B system. I concur mildly and am willing to accept a better proposal, but none has been made. Recently I have used the addition of the letter g (that is, Ag) which seems to me to make the symbol more acceptable. Using this symbol the genotypes of the three phenotypes would be represented as follows:

Blood Group or Phenotype	Genotype
M	Ag^M Ag^M
MN	Ag^M Ag^N
N	Ag^N Ag^N

The symbols for alleles responsible for subgroups of M and N can readily be developed by adding numbers to the superscripts.

The proposed Rh-Hr phenotype and gene symbols probably are the ones which have resulted in the greatest amount of disagreement and the ones which should receive immediate attention.

I shall not attempt to develop the entire history which relates to the discovery of the Rh-Hr blood variations. Suffice it to point out that at least three pairs of Rh-Hr antigens are now known. I shall give general credit for their discovery to Landsteiner and Wiener, Levine, Race, Mourant, and Diamond.

The three pairs of Rh-Hr antigens are universally recognized by a set of symbols which may be shown in the following paired order:

Rh′ and Hr′
Rh_o and Hr_o
Rh″ and Hr″

The paired relationship is based on the fact, that with respect to each pair, either member may exist by itself in the blood of a given individual or in combination with the other one. Furthermore, each possible blood group, with respect to a given pair, may exist in combination with each possible combination of the two other pairs. Hence 27 Rh-Hr blood types are possible. At the very outset of the discovery of the first Rh antigens Wiener gave abbreviated names to the known Rh blood types. As more and more antigens were detected he coined abbreviated names for the new possible blood types. Sometimes he used an old name with a new meaning. All of this would have been appropriate had he been successful in developing a system of names which would have been descriptive, easily learned, and readily presented. Many are of the opinion that he has failed to do this.

A few years ago Fisher and Race suggested the use of the letters C,c,D,d, E and e for the antigens Rh′, Hr′, Rh_0, Hr_0, Rh″ and Hr″ respectively. In accordance with this suggestion a blood type containing the antigens Rh′, Rh_o, and Rh″ would be listed as CDE, and one containing antigens Rh′, Hr_0 and Hr″ as Cde. In my opinion this letter system of names for blood types, proposed by Fisher and Race, is far superior to that devised and strongly advocated by Wiener. I might point out parenthetically that the Fisher-Race system of names is almost universally used in Europe and is also widely accepted in this country. Since it is such a simple and descriptive system I should like to urge its universal adoption. However, I should like to see the symbol Rh presented in front of the letters for the antigens, especially when a single Rh-Hr blood type is listed. In accordance with this suggestion the 27 blood types would be represented as follows:

Blood type using the original names of the antigens.	Recommended names.
1. Rh′ Rh_o Rh″	Rh CDE
2. Rh′ Rh_o Rh″ Hr″	Rh CDEe
3. Rh′ Rh_o Hr″	Rh CDe
4. Rh′ Rh_o Hr_o Rh″	Rh CDdE
5. Rh′ Rh_o Hr_o Rh″ Hr″	Rh CDdEe
6. Rh′ Rh_o Hr_o Hr″	Rh CDde
7. Rh′ Hr_o Rh″	Rh CdE
8. Rh′ Hr_o Rh″ Hr″	Rh CdEe
9. Rh′ Hr_o Hr″	Rh Cde
10. Rh′ Hr′ Rh_o Rh″	Rh CcDE
11. Rh′ Hr′ Rh_o Rh″ Hr″	Rh CcDEe
12. Rh′ Hr′ Rh_o Hr″	Rh CcDe
13. Rh′ Hr′ Rh_o Hr_o Rh″	Rh CcDdE
14. Rh′ Hr′ Rh_o Hr_o Rh″ Hr″	Rh CcDdEe
15. Rh′ Hr′ Rh_o Hr_o Hr″	Rh CcDde
16. Rh′ Hr′ Hr_o Rh″	Rh CcdE
17. Rh′ Hr′ Hr_o Rh″ Hr″	Rh CcdEe
18. Rh′ Hr′ Hr_o Hr″	Rh Ccde
19. Hr′ Rh_o Rh″	Rh cDE
20. Hr′ Rh_o Rh″ Hr″	Rh cDEe
21. Hr′ Rh_o Hr″	Rh cDe
22. Hr′ Rh_o Hr_o Rh″	Rh cDdE
23. Hr′ Rh_o Hr_o Rh″ Hr″	Rh cDdEe
24. Hr′ Rh_o Hr_o Hr″	Rh cDde
25. Hr′ Hr_o Rh″	Rh cdE
26. Hr′ Hr_o Rh″ Hr″	Rh cdEe
27. Hr′ Hr_o Hr″	Rh cde

I shall not list the names of the 27 Rh-Hr blood types proposed by Wiener, primarily because I am not certain how he would designate some of the combinations. A partial list may be found in one or more of his recent publications.

The symbolism of the genes responsible for the 27 Rh-Hr blood types is also in a state of confusion, partly because of some question regarding the mode of inheritance of these blood types, but primarily because of the disagreement which exists regarding the appropriateness of the proposed symbols.

Landsteiner and Wiener were the first to demonstrate inheritance of Rh blood variations. They concluded in 1941 that a single pair of autosomal alleles, which they called *Rh* and *rh*, would account for the then known Rh positive and Rh negative blood types. As more and more antigens were discovered Wiener concluded that additional alleles were involved. He now postulates eight alleles with no dominance relationships. Such alleles, in combinations of two, give 36

genotypes but only 27 phenotypes.

In 1944 Fisher and Race proposed a different mode of inheritance for the Rh blood complex. They postulated that three closely linked loci are involved and that each is represented by a pair of alleles with no dominance relationship. They called these three pairs of alleles *C* and *c*, *D* and *d*, and *E* and *e*. Although Fisher and Race have not demonstrated crossing over between the three loci they have assumed that this process occurred in the past, and that accordingly eight gene combinations exist on present day chromosomes of the human species. These eight possible combinations, using their symbolism, are, of course, *CDE*, *CDe*, *CdE*, *Cde*, *cDE*, *cDe*, *cdE*, and *cde*.

Thus two quite distinct hypotheses have been proposed for the inheritance of the observed Rh-Hr blood variations. The one may be referred to as the eight allele hypothesis of Wiener, and the other as the three locus hypothesis of Fisher and Race.

Now it is relatively difficult to decide in favor of the one or the other of these two proposed genetic hypotheses. One convincing way is to find direct evidence of crossing over between the three assumed loci. If such crossing over can be demonstrated on a fairly large scale then the three locus hypothesis of Fisher and Race must be favored. (A single incident of assumed crossing over would not be sufficient evidence in favor of the three locus hypothesis, because it might represent a mutation rather than a crossover.)

Although the three locus hypothesis of Fisher and Race is attractive, it is my opinion that until crossing over has been demonstrated, or until other convincing evidence is presented in favor of this hypothesis, the eight allele hypothesis of Wiener must be defended. If linkage at the present time is complete, as Fisher has stated that it may be, then there is no justification for assuming the existence of three loci. A gene is what behaves as a unit in heredity.

Although I will defend the eight allele hypothesis of Wiener until convincing evidence is presented in favor of some other hypothesis, I do not consider acceptable the allelic symbols which Wiener has proposed. The reasons for my objection I shall develop later.

I have already argued in favor of the Fisher-Race system of letters for the names of the antigens. Consistent with this argument, I should like to propose a *CDE* system of names for the eight alleles. It consists of *Rh* as the basic locus symbol with the appropriate letters as superscripts. This system is descriptive, easily learned, readily presented and in conformity with recommended genetic rules. The eight proposed allelic symbols together with the corresponding names used by Wiener are shown below.

Proposed allelic Symbols	*Corresponding Wiener Symbols* (1947)
Rh^{CDE}	R^z
Rh^{CDe}	R^1
Rh^{CdE}	R^y
Rh^{Cde}	r'
Rh^{cDE}	R^2
Rh^{cDe}	R^o
Rh^{cdE}	r''
Rh^{cde}	r

Before discussing the advantage of the proposed *CDE* system of gene symbols over the Wiener system, I should like to argue for the retention of *Rh* as the basic Rh locus symbol, rather than using only the single letter *R*. This is not an argument against Wiener's gene symbolism, because the single letter *R* could be used in the *CDE* system. The argument which I wish to advance is that the double letter symbol *Rh* is very descriptive of the characteristic involved and for this reason should not be dropped. If only 26 gene loci existed on human chromosomes, one migth argue for the smaller number of letters in a symbol; but since hundreds of human loci exist and since there are only 26 letters in the alphabet, many human loci will have to be represented by double letters, or perhaps even by three letters. Thus, there appears to be no reason for ambiguity in dropping the letter *h* when the symbol *Rh* is not cumbersome.

The arguments against the Wiener

gene symbols are that they are not descriptive, not easy to learn, not easy to present clearly in longhand or print, and that they are not in conformity with good genetic usage. It is true that if one memorizes the meaning of all of Wiener's abbreviations and uses them constantly over a period of time, one might develop a certain facility with them. However, at a glance the name of any given allele does not convey directly any specific action of the gene concerned. Furthermore, when written in longhand on paper or on the blackboard, the prime superscript and number 1 superscript are not easily distinguished. Finally, it may be pointed out that the use of some capital letters and some lower case letters for gene symbols is not in conformity with genetic usage since dominance and recessive relationships do not exist in this instance.

I should also like to point out that if it should prove to be true that three loci are involved rather than a single locus, Wiener would have considerable difficulty in transforming his symbolism to conform to the three locus hypothesis, whereas the CDE system could readily be modified to conform. For example, if three loci are shown to be involved, the three pairs of genes could be called Rh^C and Rh^c, Rh^D and Rh^d, Rh^E and Rh^e. I must admit that it is true that this usage of a single basic gene symbol (*Rh*) for three different loci would not be based on genetic precedents, but since, in this instance, the genes of all three loci would concern the same general characteristic, and probably derived from the same source, the deviation from custom would not be marked. As a matter of fact, valid argument from a purely genetic point of view could easily be developed in favor of the adoption of this system.

In conclusion I should like to urge strongly not only the universal adoption of single sets of names and symbols for human phenotype and gene symbols, but also the adoption of names and symbols which are descriptive, easily learned, readily presented and in conformity with recommended genetic rules. I am confident that the science of human genetics would profit immeasurably if these suggestions were invariably followed.

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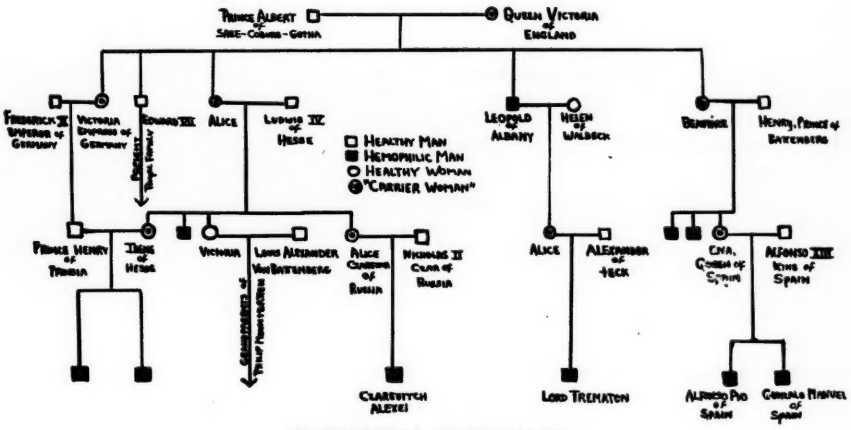
HEMOPHILIA, "THE ROYAL DISEASE"

And the British Royal Family

HUGO ILTIS

Mendel Museum, Fredericksburg, Va.



HEMOPHILIA IN ROYALTY

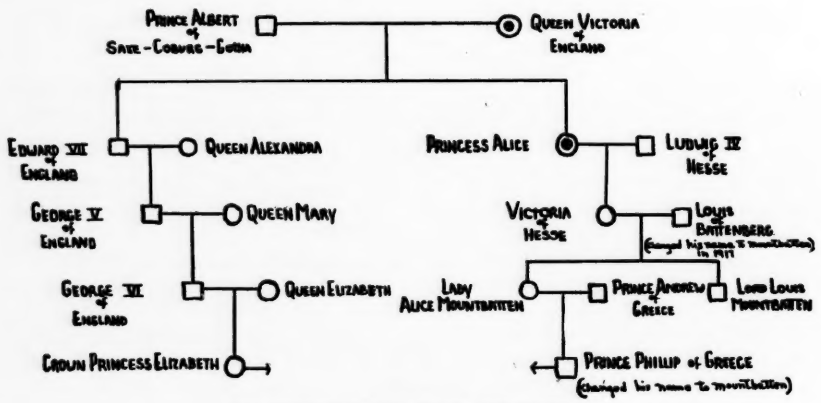
Figure 7

One, or possibly two, of Queen Victoria's sons suffered from hemophilia and through two of her daughters "the royal disease" was carried into the Russian and Spanish ruling families. By contributing to the hold of Rasputin on the Czarina, and by endangering the succession in Spain, hemophilia has had a considerable influence on recent European history.

THE characteristic sex-linked inheritance of hemophilia was first described scientifically by the American author, John C. Otto in 1803. Since genetics became a science the reasons for this peculiar pattern of inheritance have been thoroughly studied.

A special interest and even some historical importance became attached to hemophilia by its frequent appearance among the royal families of Europe during the last two generations. The last Czarevitch of Russia, Alexei, and two sons of Alfonso XIII, the last king of Spain, suffered from hemophilia. The disastrous influence of the fanatical monk Rasputin upon the Czarina and Czar Nicholas II came from his alleged hypnotic control of the disease in the little Alexei. It is not impossible that the affliction of the crown princes of these ruling families with the hereditary "royal disease," as it was called, played a significant part in the overthrow of these two dynasties. The recent marriage of two closely related members of the hemophilic strain of these families, Princess Elizabeth and Philip Mountbatten, raises the question of whether there is any danger that the fateful gene may reappear among any of their children or descendants.

During the last ten years several scientists (Gunn,[3] and Haldane,[4] etc.) have tried to trace back to its origin the abnormal gene exhibited in the Russian and the Spanish royal families, and in several other princely families in Europe. As far as existing evidence is conclusive, Queen Victoria of



NO HEMOPHILIC SONS EXPECTED

Figure 8

Both Princess Elizabeth and her consort are descended from Queen Victoria. But the peculiar inheritance of sex-linked traits makes it impossible that the children of this marriage can have or transmit the hemophilic gene which has blighted the lives of a number of Victoria's male descendants.

England was the first carrier of the morbid gene. Ten of Victoria's male descendants have suffered from hemophilia and seven of her female descendants have been proved to be carriers of the gene.

One (or possibly two) of Victoria's four sons were hemophilic and two (or three) of her five daughters were "carriers" and transmitted the gene by marriage into several royal and princely families of Europe. Princess Victoria, Queen Victoria's oldest daughter, was married to Emperor Frederick of Germany. It is not certain whether she was a carrier. But it is possible, since Haldane points out that one of her sons, a younger brother of Kaiser Wilhelm, died as a child from bleeding.[5]

Queen Victoria's eldest son, King Edward VII, was not a hemophiliac and could not have transmitted hemophilia to any of his descendants. *The fact that Queen Victoria was a carrier does not imply that there is any trace of the disease in the present British Royal Family.* Another of Queen Victoria's sons, Alfred, Duke of Edinburg, died at 56 years of age. He was not hemophilic. Arthur, Duke of Connought, like Edward VII, was free of the gene.

Leopold, Duke of Albany, the youngest of Queen Victoria's sons, was hemophilic. He survived, however, and married Helen, Princess of Waldeck. His son became the Duke of Sachsen-Coburg and later on, an "Obergruppenfuehrer" under Hitler. Helen's daughter, Princess Alice, who married Alexander, Prince of Teck, was a carrier and transmitted the disease to her son, who lost his life by bleeding after an auto accident.

Of Queen Victoria's daughters at least two, Princess Alice and Princess Beatrice, carried the gene of hemophilia to the next generation. Princess Alice married Ludwig IV, Grand Duke of Hesse. Of her sons, the eldest, the Grand Duke Ernst Ludwig, escaped the disease, while a younger son (Prince Friedrich) is said to have died of hemophilia. Of her daughters, the eldest, Irene, married Henry, Prince of Prussia, a younger brother of Kaiser Wilhelm. Two of her sons are said to have been afflicted[2] and therefore she has been considered as a carrier. The 1938 *Almanac de Gotha,* however, reports two sons, the Princes Waldemar, born 1889, and Sigismund,

born 1896, both still living, both married, the latter with two children. That does not look like hemophilia. Victoria, the second daughter, was married to Prince Louis of Battenberg and did not transmit hemophilia. Princess Elizabeth, her third daughter, married Grand Duke Serge of Russia and had no children. It is therefore uncertain whether she was a carrier. The youngest daughter, Princess Alice, married Czar Nicholas II of Russia and became Czarina Alexandra. Her son, Czarevitch Alexei, was a typical hemophilic. Fear for his life guided the actions of the Czarina. The sinister influence of the monk Rasputin, who claimed to be able to stop the bleeding, thus finds its explanation. Some of the four daughters of Czarina Alexandra were probably carriers. But all of them with their parents and their brother perished during the Russian revolution.

Queen Victoria's youngest daughter, Princess Beatrice, was also a carrier. She was married to Henry, Prince of Battenberg, and transmitted the hemophilia gene to two sons, who died as children, and to her daughter, Princess Victoria Eugenia, who later became the wife of Alfonso XIII, King of Spain. She transmitted the disease to two of her sons. Prince Alfonso Pio died in an automobile accident in Florida in 1938; the younger son, Prince Gonzalo Manuel, died from bleeding as a child.

Origin of the Mutation

The question arises from where and how Queen Victoria acquired the fateful gene of hemophilia. A definite answer to this question will be difficult if not impossible. Queen Victoria's father, Edward Duke of Kent, was not a bleeder and therefore could not have transmtited the gene to his daughter. Her mother, the Duchess of Kent, was born as a Princess of Saxe-Coburg in 1786. She had two daughters by a former marriage who do not seem to have carried the gene. She herself was one of a large family, ten in all, five sons and five daughters. Two of these ten children died young and one was stillborn. Hemophilia as a disease was not recognized at that time. The possibility is not excluded, although there is no record preserved, that some or all of the male children may have been hemophiliacs. Going back one more generation, the gene could not have been brought in by Queen Victoria's grandfather, the Duke of Saxe-Coburg, since he was not a bleeder. The gene could have been transmitted only by her grandmother, Princess Augusta, born Princess of Reuss. She, too, was a member of a large family and here again three of her six brothers and sisters died young. The record gives no evidence that any of them were bleeders, though this remains a possibility.

Haldane[4] suggests an alternative possibility that the gene for hemophilia may have originated by mutation in Queen Victoria herself. Such instances of spontaneous mutation are well known in animals and plants, and Haldane is convinced that the hemophilia locus mutates rather frequently in human beings.

The practical importance of genetics in human affairs is brought into sharp focus by the oblique indications from London that Elizabeth is expecting an heir. Not only are Elizabeth and Philip both great-great-grandchildren of Victoria, but they are related by several other lines of descent.[6] In the presence of a morbid gene like hemophilia, such a marriage might seem hazardous, especially when a male heir is a matter of primary importance. In this particular case, however, modern science based upon our knowledge of the laws of inheritance of a sex-linked character removes any reasons for any apprehension with respect to hemophilia.

Crown Princess Elizabeth traces to Queen Victoria through three generations of male descendants (King Edward VII, King George V, King George VI). Since none of them was hemophilic, we know that she cannot have inherited this fateful gene from Queen Victoria. Prince Philip Mountbatten is the great-grandson of Princess Alice of Hesse, who was a carrier and who transmitted the disease to one son and to two of her daughters as carriers, Irene of Prussia

and Alexandra of Russia. Since Philip's relationship to Queen Victoria is through the female line, there might appear to be a possibility of transmission through him. Philip himself answers this apprehension: since he is not hemophilic, he cannot carry the gene. There is therefore no danger whatever that the fateful gene of hemophilia will be carried into the next generation through this marriage.

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A TEXT FOR A COURSE ON HABROBRACON

DR. Martin of the University of Pittsburgh has produced an attractive little book summarizing the published work on the parasitic wasp now well-known to geneticists. The author's aim has been to write a book "primarily for use in course work" and in that he has notably succeeded. After introductory material including life history and culture methods (21 pp.), mutant types are listed and described (47 pp.). Methods of recording data are briefly and clearly given for the benefit of the student. Sex conditions and the complementary type of sex determination found in *Habrobracon* are discussed at some length (29 pp.). The linkage map and methods of determining linkage are shown (9 pp.). Under environmental effects (19 pp.) are included influences of temperature on body and eye colors, on fecundity and on ratios of sex types. Production of lethal and of visible mutations by x-radiation of sperm and of eggs is discussed.

The 19 clear and well-reproduced illustrations in the text and the three tables in the appendix should be helpful. The bibliography (30 pp.) includes all but the most recent references concerning *Habrobracon* as well as several others that are pertinent. Entomological and genetical terms are brought together and clearly defined to make up a unique glossary pertaining to this type of material. A few minor errors do not materially mar the book for its intended purpose.

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*MARTIN, ALBERT, JR. *An Introduction to the Genetics of Habrobracon juglandis (Ashmead).* xii + 205 pp. $3.50. The Hobson Book Press, New York. 1947.

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Dr. Lush to Survey British Genetics

Dr. Jay L. Lush, Iowa State College, is on temporary duty until July 31 with the U. S. State Department's Mission on Science and Technology with headquarters at the U. S. Embassy in London. He will study and report on research in genetics, animal breeding, and related fields of animal production in the British Isles. Inquiries about topics in this field on which information is desired will be welcomed. In view of the short time Dr. Lush is in London, these should be forwarded as soon as possible.

CYTOLOGICAL EFFECTS OF ALPHA-NAPHTHALENE ACETIC ACID

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IN 1938 Greenleaf[6] induced the formation of polyploid shoots in several species and hybrids of *Nicotiana* by applying a lanolin paste containing indole-3-acetic acid to the cut surface of decapitated stems. He did not discuss the mechanism by which the polyploid condition may have arisen. Four years later Beal[1] stated that there was no evidence from experiments on bean, lily and tomato that indole-acetic acid induces polyploidy. Since decapitated tomato stems give rise to polyploid shoots without treatment, Beal suggested that in Greenleaf's experiments the indole-acetic acid may have stimulated the callus growth while the polyploid shoots were caused by the same factors which produce them in tomato. What these factors might be and how they operate in producing polyploidy was not discussed by Beal.

In 1939 Levan[7] studied the cytological effects of four growth promoting substances including naphthalene acetic acid on *Allium*. He concluded that these substances stimulate mitosis in the meristematic region of the root and induce polyploidy in the large differentiated cortical cells by the following mechanism. The primary effect is an increase in the volume of the cell which is followed by a doubling of the chromosomes within the resting nucleus. In 1943 Carlton[4] treated *Allium, Tulipa,* and *Narcissus* with alpha-naphthalene acetic acid and several other growth promoting substances. In *Tulipa* and *Narcissus* no polyploid divisions were found and the few observed in *Allium* were not more numerous than the number found in untreated material.

In 1941 Dermen[5] treated the internodes of bean plants with naphthalene acetic acid in lanolin paste and stated that polyploidy was induced in the differentiated cortical cells but not in the active meristem. He found 4*n*, 8*n* and 16*n* divisions and presented evidence that the polyploid condition was the result of double chromosome reproduction in the resting nucleus. In view of this diversity of opinion as to the possible polyploid-inducing activity of alpha-naphthalene acetic acid a reinvestigation of the cytological effects of this substance was undertaken. A preliminary report of our results[2] was presented at the Boston meeting of the A.A.A.S. in 1946.

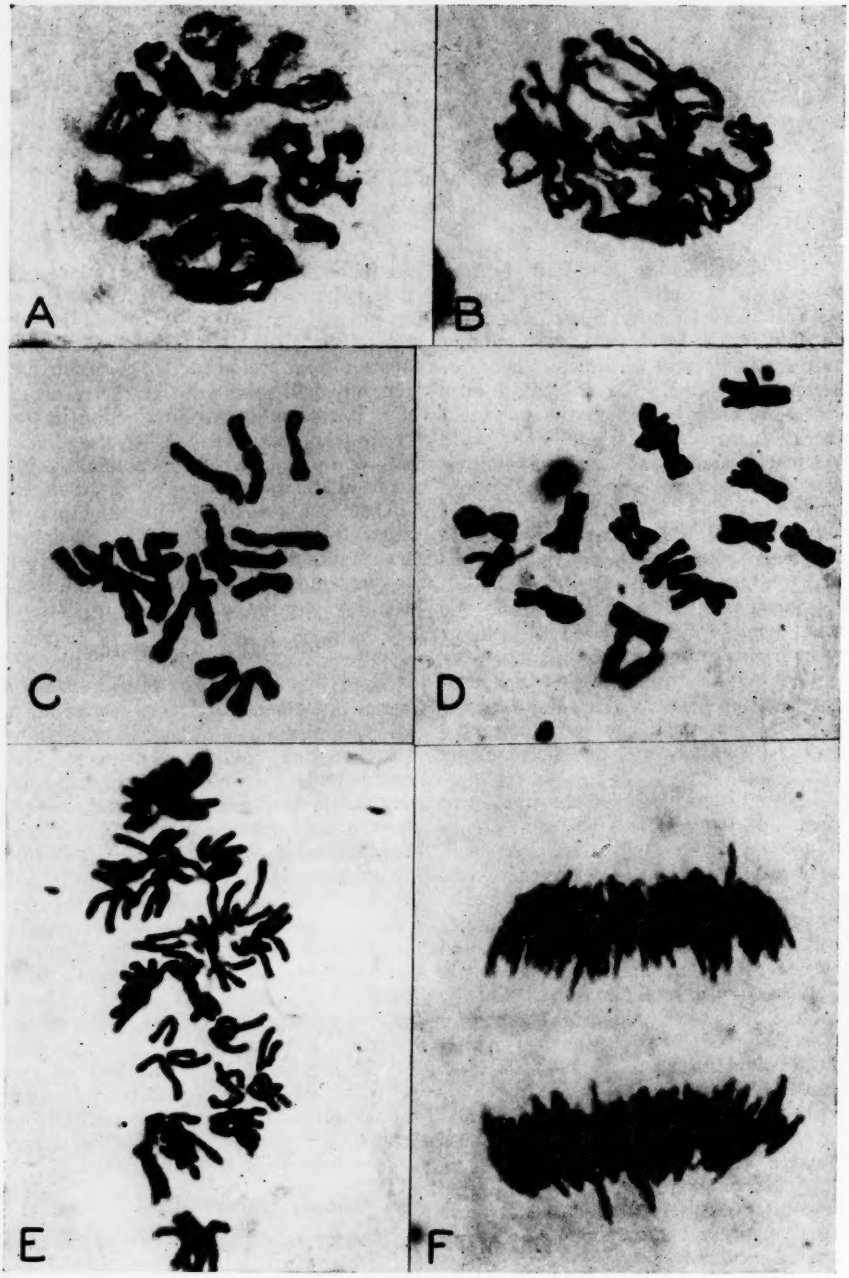
Material and Methods

Root tips from bulbs of *Allium cepa* were immersed for four hours in a 0.0005 per cent solution of alpha-naphthalene acetic acid. Since this acid is not soluble in water a few drops of N/KOH were added; hence the agent was a tap water solution of the salt naphthalene acetate. After the four-hour treatment some roots were cut and fixed immediately and others after four, eight, twelve, twenty-four and forty-eight hours of recovery in tap water. The tips were fixed in absolute alcohol and glacial acetic acid (3:1) and smear preparations were made and stained with aceto-orcein. Sectional material was also prepared for accurate localization of the effects. *Allium cepa* was used because of its suitability for such tests and because Carlton and Levan obtained different results with this material.

Observations

The effect of naphthalene acetic acid on the cells of the undifferentiated meristem is very different from its effect on those of the region of differentiation.

*Aided by a grant from the National Advisory Cancer Council.



TETRAPLOID CELLS STIMULATED TO DIVISION BY NAPHTHALENE ACETIC ACID

Figure 9

A and B—Prophase. *C*—Prometaphase. *D*—Metaphase. *E*—Very late metaphase. *F*—Anaphase. All photomicrographs are of aceto-orcein smear preparations of the region of differentiation of roots of *Allium cepa*. Magnification 1000x.

Root tips treated for four hours with a 0.0005 per cent solution and fixed immediately showed that this substance had little effect on meristematic cells undergoing mitosis. The division of the SA-region (spindle attachment region) was delayed and the chromosomes were held at metaphase and became more highly contracted than usual. The spindle was not affected and anaphase separation followed normally. Resting cells in the apical meristem were not stimulated to divide. It was suggested that such a stimulation would have taken place if a much lower concentration of the acid had been used. Accordingly experiments were conducted in which the concentration was reduced to one part per million and one part per ten million but in neither case was any stimulation of mitosis noted in the meristematic cells of the root tip. It may be that the "stimulation" of mitoses attributed to this substance is actually an illusion produced by the twenty-four hour inhibitory period. Treatment with naphthalene acetic acid hinders meristematic resting cells from entering division for twenty-four hours from the beginning of treatment. At the end of this period more cells than usual are ready for mitosis and begin to divide so that observations made at this time could easily be misinterpreted as indicating a stimulation effect. Our observations, therefore, indicate that in the undifferentiated meristem naphthalene acetic acid neither stimulates mitosis nor induces polyploidy.

The most pronounced effect of this substance was produced in the region of differentiation. After four hours of treatment and twelve hours of recovery many cells of the pericycle were found undergoing mitosis. The divisions were all diploid and were much more numerous than those in untreated roots. After forty-eight hours of recovery in tap water many of the large cells of the cortex entered division and were found to be tetraploid.

The tetraploid divisions have several distinctive cytological features as illustrated in Figure 9, *A* to *F*. From prophase to prometaphase the thirty-two chromosomes are in sixteen units, each unit consisting of two chromosomes united at an undivided SA-region. This configuration has been described in a number of cases of polysomaty and of naturally occurring polyploidy where it constitutes evidence (1) for the origin of the polyploid condition by double chromosome reproduction in the resting nucleus and (2) that the divisions showing this configuration are the first mitotic divisions after the tetraploid condition was attained. Only in a very broad and inaccurate sense do these divisions resemble meiotic divisions. The paired association is due to reduplication without separation, not to active pairing. As Lorz[8] has expressed it such a "paired" condition is not due to *association* but to the absence of *dissociation*. There is relational coiling of the paired chromosomes but no chiasmata are present. The most illuminating characteristic is the single undivided SA-region holding the two chromosomes together up to metaphase instead of the two widely diverging or repelling SA-regions of a meiotic bivalent.

Discussion

There are two possible explanations of the origin of the tetraploid cortical cells. Tetraploidy may have been induced by the treatment or the cells may have been tetraploid previous to the treatment and merely stimulated to divide by the naphthalene acetic acid. The second possibility is more probable for the following reasons.

Cases of the normal occurrence of polyploid cells in diploid plants have been reported for many species including *Allium*.[3] Among the different species showing polysomaty (see Lorz[8]) some have cells which divide several times as polyploids and reach high degrees of polyploidy, e.g., the octoploid cells of *Spinacia* which undergo at least three divisions as polyploids, while other species have cells which do not go beyond a $4n$ condition and usually divide

only once as tetraploids before becoming differentiated. It is very probable that still other species have some cells which become tetraploid by double chromosome reproduction in the resting nucleus and then undergo differentiation without dividing even once as tetraploids. Slight variations in the time at which mitotic activity ceases and differentiation occurs would account for the many reported cases of rare 4*n* dividing cells in diploid species.

If cell size is taken into account together with the known occurrence of 4*n* cells in the shoot of *Allium* and in the root and shoot of other species, it does not appear improbable that the large cortical cells of *Allium* roots are tetraploid. Furthermore naphthalene acetic acid stimulates many small cells of the differentiated region to undergo repeated mitosis without causing them to become polyploid.

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GENETICS AND PATHOLOGY OF Rh BLOOD

EVERY so often a concept appears on the medical horizon which not only unifies and clarifies previously vaguely interrelated phenomena, but opens up new vistas of experimentation and analysis. Such a unifying concept is the maternal-fetal interaction of immune iso-antibodies, as typified by the Rh blood factors. Within the short span of less than a decade more than a thousand papers have been written on this subject. The literature is widely scattered in medical and genetic journals, and the problem has become increasingly more inclusive and more complex.

A thorough and comprehensive summary of this involved subject has been sorely needed. Such a volume is now available.* Dr. Edith L. Potter brings to the preparation of this book a long experience in the fields of obstetrical practice and diseases of the fetus and the newborn infant. Moreover, she has had the opportunity to observe at first hand many thousand births, several thousand post mortem examinations of fetuses and infants, and many hundred cases of hemolytic disease. The result is a book for which there can be nothing but praise. Dr. Potter shows a clear understanding, not only of the medical problems involved, but of the genetic problems as well. The Rh problem is fundamentally one of medical genetics, and an understanding of both fields is essential to its ultimate solution.

Following a general survey of antigens, antibodies and the human blood groups, the discovery of the Rh factors is traced through the parallel investigations of Levine, Stetson and their coworkers, and of Landsteiner and Wiener. The immunology and the genetics of the Rh antigens and antibodies are outlined in a comprehensive well-written chapter. A short but complete chapter is devoted to Rh and transfusion reactions.

In a long chapter of 160 pages hemolytic disease is thoroughly treated. Stillborn fetuses without generalized edema, infants with generalized edema, infants with severe anemia but little edema, in-

**Rh*, by Edith L. Potter, M.D. xvi + 344 pp. Chicago, the Yearbook Publishers, Inc. 1947. $5.50.

fants with progressive anemia, and infants with little jaundice and slowly progressive anemia are all comprehensively discussed. A series of fine illustrations adds much to the value of this chapter. The clinical and differential diagnosis of hemolytic disease is carefully outlined, and the complications, sequellae, prevention and treatment of the condition are extensively treated. The final section of this long chapter is devoted to a complete and well-illustrated description of post mortem examinations.

The last chapter deals clearly and in specific detail with the technics for determination of Rh status of red blood cells and presence of Rh antibodies in the blood. This chapter, too, is well illustrated with excellent photomicrographs and diagrams.

A bibliography of 794 titles and a good index complete the book. The paper is excellent, and the general make-up of the volume is pleasing. An interesting and valuable innovation is the very first chapter, which is a short general survey of the material contained in the rest of the volume. The Wiener terminology is used throughout, although comparison and reference is made to the CDE and other systems of nomenclature.

This volume will rank with the finest contributions to the literature of medicine and of medical genetics.

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More on The Physique-Temperament Correlation

WE are interested in the article written by Dr. Keeler in the September 1947 issue of the JOURNAL OF HEREDITY, titled "Coat Color, Physique and Temperament." In this connection, we determined the correlation between hair color and scholastic ability. This study was carried on with seniors of this year in different schools of Oregon State College.* Those with medium brown to black hair were classified under "dark," those with light brown and blond hair were included under "blond," and "red" included sandy and red headed persons. The results are shown in the following table.

ITEMS	DARK		BLOND		RED		TOTAL	
	No. of Cases	GPA†	No. of Cases	GPA	No. of Cases	GPA	No. of Cases	GPA
Men	277	2.73	141	2.69	8	2.58	426	2.72
Women	106	2.90	69	2.88	3	2.59	178	2.89
Total	383	2.79	210	2.75	11	2.58	604	2.77

It is interesting to note that, though not statistically significant, women were better than men and that about the same trend existed in both cases of men and women; dark haired ones being the best, followed by blondes, and then red heads last. Although the number of red headed persons was very small, it may be more reliable than the number itself indicates since the GPA is almost the same for both men and women. In studying the difference between the three different classes, it was found to be statistically non-significant. ($\chi^2 = 5.27$ or $P = 0.05$ over.)

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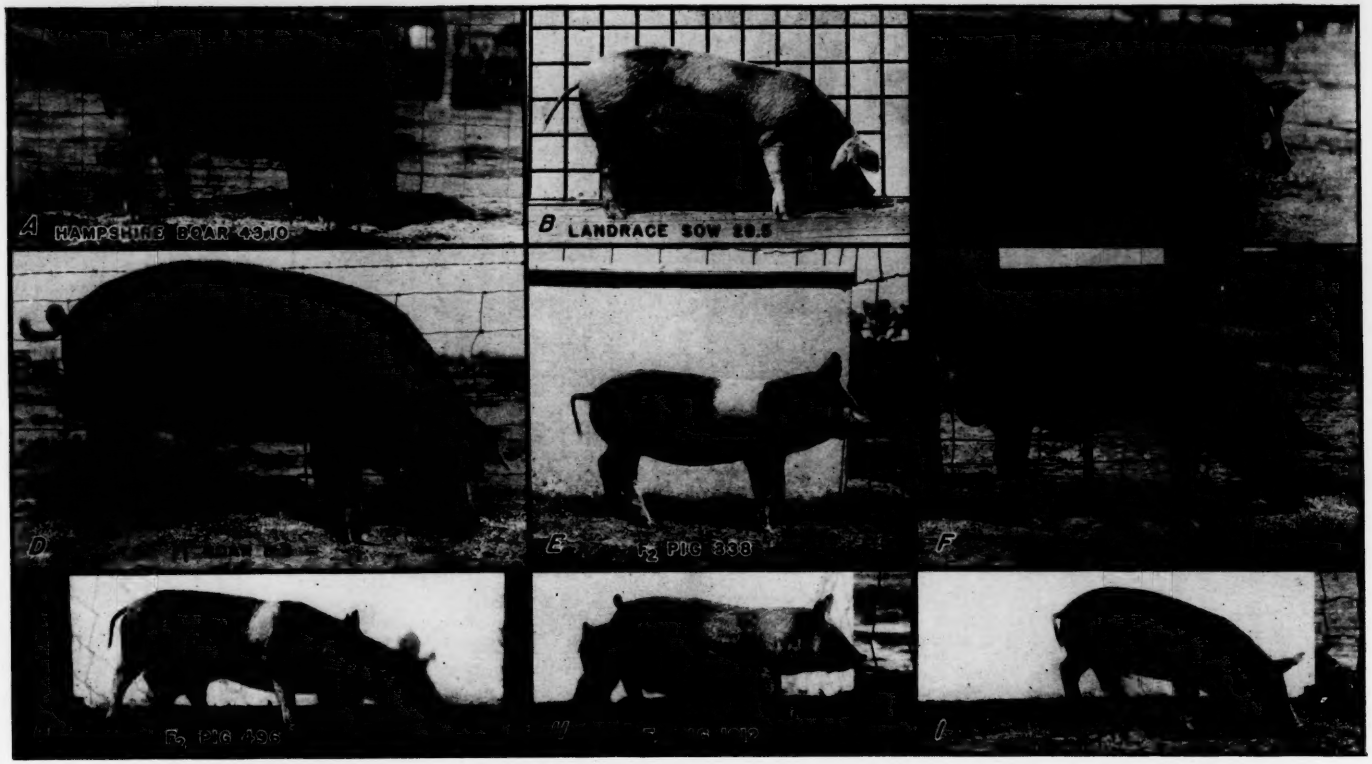
*GPA = Grade Point Average, maximum 4.00 points. Grade A = 4 points, B = 3 points, C = 2 points, D = 1 point, F = 0 points.

†The data were secured through the courtesies of: Mr. W. M. Langan, Agricultural Student Personnel Advisor; Dean Clifford E. Maser, Head, Division of Business and Industry; Dean Ava B. Milam, Dean of the School of Home Economics; Dean F. A. Gilfillan, Dean of the School of Science; Dean E. G. Crossen, Dean of the School of Pharmacy; Dean J. R. Jewell, Dean of the School of Education; Dean G. W. Gleeson, Dean of the School of Engineering and Industrial Arts.



FIRST AND SECOND GENERATION HYBRIDS OF LANDRACE AND HAMPSHIRE

Figure 10

A—One of the two Hampshire boars used in the original cross. This boar, like all other Hampshires used in the experiment was entirely black. *B*—Sow 28.5, one of the two Landrace sows which produced both whites and blue roans in F_1. *C*—One of the blue roan F_1's out of sow 28.5 by boar 43.10 (shown in *A*). Note close intermingling of black and white hairs with longitudinal stripes. *D*—A blue roan F_1 boar out of a sister of sow 28.5 by Hampshire boar 41.8. *E*, *F*, *G*, *H*, and *I*—F_2 pigs out of sow 9.10 (shown in *C*) by boar 8.5 (shown in *D*), illustrating the five principal color varieties obtained in later generations. Pig in *E* was blue roan with a Hampshire-like belt. Boar shown in *F* was solid black. Pigs in *G*, *H*, and *I* were red roan, white and red with black spots, respectively.

INHERITANCE OF COAT COLOR IN SWINE

VII. Results of Landrace by Hampshire Crosses*

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PREVIOUS papers by the author[1,2,3,4,5] have shown that the white hair color of the Landrace and Yorkshire is due to a dominant inhibitor (I) and that its recessive allele (i), carried by the Poland China, Berkshire, Large Black and Duroc-Jersey breeds, is necessary for the production of either black or red color. It was also shown that the Landrace and Yorkshire possess the same black spotting gene (E^p) as the Poland China and Berkshire and that its dominant allele (E) results in complete extension of black as found in the Large Black. The absence of black in the Duroc-Jersey in turn was found to be conditioned by a simple recessive to black spotting, indicating that complete extension of black (E), partial extension of black (E^p) and complete restriction of black (e) form a series of triple alleles with dominance in that order. The Landrace and Yorkshire were thus represented by the formula E^pE^pII, the Poland China and Berkshire by E^pE^pii, the Large Black by $EEii$, and the Duroc-Jersey by $eeii$.

As regards the inheritance of the Duroc-Jersey's red color, the evidence supported the conclusion that the Landrace, Yorkshire, Poland China and Berkshire probably have two or three cumulative partially dominant genes for dilution of red (D_1D_2–) while the Duroc-Jersey has red intensity factors (d_1d_2–), some of which probably also occur in the Large Black. The purpose of this paper, the last one in this series, is to describe a previously unreported gene in the Landrace and to discuss its relation to certain other color genes differentiating the Landrace from the Hampshire.

The Hampshires used in the experimental crosses came from a group of eight animals of which four were obtained from the college herd of the University of Nebraska and four from a commercial breeder in Ohio. The Nebraska animals came, according to Professor Wm. J. Loeffel, from matings between belted individuals, but they, as well as those received from Ohio, were entirely black. The Landrace animals were all derived from the same source as those used in the crosses reported in previous articles in this series. Their hair was uniformly white, as was the skin, with the exception of a few black spots in some animals.

Effects of the Gene I^d

The new allele of the Landrace inhibitor, I, to which the symbol I^d (dilution) has been given, is intermediate between I and i in its action in animals possessing the gene for self black (E). Such animals (I^dI^d) show a blue roaning pattern very similar to that of the Sapphire hog described by McLean[6] in this JOURNAL in 1914. Roan pigs obtained in this experiment were characterized by a close intermingling of black and white hairs and also by a number of white markings. In some animals the white markings were confined to the feet, the tip of the tail and to a splash of white on the face; in others they extended over the shoul-

*Data obtained at Miles City, Montana, by the Bureau of Animal Industry, U. S. Department of Agriculture in cooperation with the Montana Agricultural Experiment Station.

†The author acknowledges his indebtedness to Mr. R. E. Hutton for recording the colors of the pigs used in this study.

ders and back, thereby giving rise to a belt which in some animals was so extensive that only the head and part of the rump showed the blue roaning pattern (see Figure 10). These findings might suggest segregation for a belting factor, but three considerations seem to refute such an interpretation. First, none of the Hampshires used as foundation animals, nor any of their purebred descendants (113 pigs), showed any trace of white hairs. Second, although some of the blacks obtained in the experiment had a little white on their feet or legs, none showed a belt. Third, in most of the blue roans the belting was much more irregular in outline than that of the belted Hampshire. It seems best, therefore, to leave open the question whether the belting observed in some of the blue roans was due to a specific belting factor or whether it was due to certain minor genes modifying the expression of the I^d gene. As to intensity of color, the blue roaning pattern graded from a deep bluish gray to a light grayish blue, barely distinguishable from white. Accordingly, there was probably some overlapping with white, but there were no indications that this introduced any serious errors. It might be added that most blue roans were born with longitudinal stripes very similar to those in the young of the wild hog. In some the striping disappeared with advancing age, but as a general rule the stripes remained fairly distinct.

Only a few matings were made to study the interaction of E^p and I^d, but all animals known to have the constitution E^p–I^d– showed a number of black skin spots which in some cases were covered with mixed black and white hairs. The spots were similar to those of some E–I– and E^p–I– whites but in no case did their distribution resemble that observed in the phenotypically black-spotted animals obtained from some of the matings. It seems probable, therefore, that in genetically black-spotted animals I^d suppresses the production of black in the same manner as its allele I. As to its effects on red, the evidence suggests that I^d dilutes red to red roan or white. However, since some of the genes for dilution of red previously reported in the Landrace undoubtedly were involved in some of the matings, it is impossible to assert that the red roans obtained in the experiment owed their appearance to the I^d gene. As most animals postulated to be E^p–I^d– were red roan in color, it is evident nevertheless that, unlike its allele I, I^d does not completely suppress the production of red. Incidentally the red roans closely resembled the blue roans in all respects except color.

Identification of I^d

The presence of the gene I^d was noted first in the F_1 offspring* of two Hampshire boars (41.8, 43.10) by two Landrace sisters (28.3, 28.5). These matings produced 23 pigs, of which 11 were white and 12 blue roan. By three other Landrace sows the same boars sired only white pigs, 25 in all. Matings of two Hampshire sows with a Landrace boar produced 17 pigs, also all white. Two of the seven P_1 matings were thus found to produce both whites and blue roans in about equal numbers.

These results are consistent with the suggestion that blue roaning is due to a mutation of I to I^d, an intermediate allele of I and i which in combination with the dominant gene E (self black) reduces black to blue roan. Landrace sows 28.3 and 28.5 evidently were heterozygous for I ($E^pE^pII^d$), while the other Landrace animals were E^pE^pII. Mated to Hampshires ($EEii$) the offspring of sows 28.3 and 28.5 would thus consist of equal numbers of EE^pIi (white) and EE^pI^di (blue roan). The offspring of the other Landrace × Hampshire matings would be EE^pIi (white).

One of the Landrace sows (28.5) which had produced blue roan progeny later was mated to a blue roan F_1 boar

*In this discussion the genetic terminology—P_1, F_1, F_2, and backcross, is used loosely to describe the relationship of the individuals in question to the parent breeds, even though the terminology is incorrect in a strict sense with respect to the alleles I and I^d under discussion.

(8.5) out of sow 28.3. This mating gave 11 whites and 3 blue roans. One of the Landrace sows which had produced only white F_1's again produced only whites (12 pigs) when mated to a blue roan F_1 boar (9.5) out of sow 28.5. Of three additional Landrace sows which were mated to these two F_1 boars, two produced only whites (7 and 13 pigs), and one produced 9 whites and 1 blue roan. The progenies of the two Landrace sows which in the above backcross proved capable of transmitting blue roaning thus consisted of 24 pigs, of which 20 were white and 4 blue roan. This result is in good accord with a dihybrid segregation indicating that in animals homozygous for the black spotting gene E^p, the gene I^d inhibits the production of black color.

It may be added here that all blue roans produced subsequently traced to one or the other of the three Landrace sows which in the above matings had blue roan offspring and that no animals not known to carry the gene I^d ever transmitted blue roaning. Since the three sows were sired by the same Landrace boar, it is reasonable to assume that the gene I^d was transmitted to them by their sire.

The results of backcrosses to the Hampshire, including those of the various matings described above, are shown in Table I. Reciprocal backcrosses of blue roan F_1's to Hampshire gave 31 blue roans and 30 blacks, but no whites. Backcrosses of white F_1 sows to one of the original Hampshire boars, on the other hand, produced 18 whites and 16 blacks, but no blue roans. Both sets of data are in excellent agreement with their respective expectancies on the basis of a 1:1 ratio. They are thus consistent with the interpretation that the difference between white and blue roaning is due to a monohybrid segregation.

Relation of I^d and i

Five blue roan F_1 sows and two blue roan F_1 boars were available for the production of F_2 individuals (see Table II). Sows 9.6 and 9.10, both sisters of boar 9.5, each produced four kinds of pigs by boar 8.5. These consisted of 50 blue roans, 5 blacks, 11 non-blacks (7 whites, 4 red roans) and 7 red roans or reds with black spots. By two of his sisters (8.7, 8.12), boar 8.5 sired only two kinds of pigs, 19 blue roans and 11 blacks. Boar 9.5, when mated to sow 8.7 and to sow 8.8, another sister of boar 8.5, likewise produced only two kinds of pigs, 45 blue roans and 18 blacks. The four last-mentioned matings thus produced 93 pigs, of which 64 were blue roan and 29 black, without any non-blacks or black-spotted individuals. This distribution is fairly close to a 3:1 ratio ($\chi^2 = 1.90$, $P = 0.17$), supporting the hypothesis that black is a simple recessive to blue roaning. This result, when considered with the fact that boar 8.5 sired four kinds of pigs when mated to sows 9.6 and 9.10, but only two kinds when mated to sows 8.7 and 8.12, suggested at once that the sows used in the two sets of matings were genetically different, and that sows 8.7, 8.8 and 8.12 (all of which failed to produce black-spotted or non-black pigs) were homozygous for the dominant self black gene E, and hence were EEI^di. For reasons to be discussed later, it is probable that boar

TABLE I. Color of offspring of Landrace × Hampshire cross, and of backcross progeny.

Parents*		Number of pigs			
		Total	White	Blue roan	Black
Hampshire × Landrace					
41.8	28.3	12	6	6	..
43.10	28.5	11	5	6	..
Total		23	11	12	..
41.8	92.6	11	11	..	..
43.10	18.9 & 26.10	14	14	..	..
Total		25	25	..	..
Landrace × Hampshire					
11.7	2 sows	17	17		..
F_1 blue roan × Landrace					
8.5	28.5	14	11	3	..
8.5	2.5	10	9	1	..
Total		24	20	4	..
9.5	18.2 & 26.10	19	19	..	..
8.5	3.3	13	13	..	..
Total		32	32	..	..
F_1 blue roan × Hampshire					
8.5	3 sows	28	..	13	15
9.5	2 sows	19	..	9	10
Total		47	..	22	25
Hampshire × F_1 blue roan					
43.10	9.8 & 8.12	14	..	9	5
Hampshire × F_1 white					
43.10	4 sows	34	18	..	16

*Male parent listed first.

9.5 also was EEI^di. Hence only three of the seven F_1's (boar 8.5, sows 9.6, 9.10) were EE^pI^di as originally suggested. These seven animals could have been produced by matings of $EEii \times EE^pII^d$, and it is reasonable to assume that the Landrace parents (28.3, 28.5) were EE^pII^d and that boar 9.5 and sows 8.7, 8.8 and 8.12 received a gene for black (E) from both parents.

The results of the matings involving sows 9.6 and 9.10 show that blue roans can transmit both black spotting and non-black (i.e. red roan or white), and that in genetically black-spotted animals (E^pE^p) the gene I^d suppresses the production of black color. That some of the black-spotted pigs had a red ground color, and that none of the non-blacks showed as dark a shade of red, suggests that the Hampshire carries red intensity genes and that I^d, in addition to suppressing the production of black, dilutes red to red roan or white. Since some of the genes for dilution of red previously reported in the Landrace undoubtedly were present in this experiment, such variations as did occur in intensity of red will not be considered in detail except in cases in which they afford a means for distinguishing between I^d and I. As regards the frequencies of the color varieties among the progenies of sows 9.6 and 9.10, the results (50 blue roan, 5 black, 11 non-black, 7 black-spotted) agree only moderately well with a 9:3:3:1 distribution ($\chi^2 = 9.29$, $P = 0.03$). There is a marked deficiency of blacks and a considerable excess of blue roans, but no basis exists for attributing the poor fit to linkage or to errors in classification.

Of the five F_1 sows referred to above, four (8.7, 8.8, 9.6 and 9.10) were progeny tested by matings with black-spotted boars which necessarily were E^pE^pii (see Table II). Sows 8.7 and 8.8 produced 8 blue roans and 15 blacks, but no non-blacks or black-spotted, supporting the previous conclusion that both were EEI^di. Sows 9.6 and 9.10, on the other hand, each produced pigs of four color varieties: 10 blue roans, 14 blacks, 9 non-blacks (8 red roans, 1 white) and 11 black-spotted (10 reds with black spots, 1 red roan with black spots). These results agree satisfactorily with expectation on the basis of a 1:1:1:1 ratio ($\chi^2 = 1.27$, $P = 0.74$). This confirms the interpretation that these sows were EE^pI^di and that I^d is independent of the E locus.

Relations of I, I^d and i

As a complement to the matings between blue roan F_1's, two white F_1 sows (103.8, 103.9) from the Landrace male $\times$ Hampshire female cross were mated to blue roan F_1 boar 8.5, while one of the two sows (103.8) also had offspring by blue roan F_1 boar 9.5 (see Table II). When mated to boar 8.5, sow 103.9 produced 52 pigs, distributed among five color varieties: white, blue roan, black,

TABLE II. Distribution of pigs produced by blue roan and by white F_1 sows.

Parents	Genotypes	Number of pigs					
		Total	White	Blue roan	Black	Red roan	Black-Spotted
F_1 blue roan $\times$ F_1 blue roan							
8.5 $\times$ 9.6 & 9.10	$EE^pI^di \times EE^pI^di$	73	7	50	5	4	7
8.5 $\times$ 8.7 & 8.12	$EE^pI^di \times EEI^di$	30	..	19	11	..	..
9.5 $\times$ 8.7 & 8.8	$EEI^di \times EEI^di$	63	..	45	18	..	..
	Total	93		64	29		..
F_1 blue roan $\times$ F_1 white							
8.5 $\times$ 103.8 & 103.9	$EE^pI^di \times EE^pIi$	69	42	15	8	3	1
9.5 $\times$ 103.8	$EEI^di \times EE^pIi$	38	19	12	7	..	..
Black-spotted* $\times$ F_1 blue roan							
141 $\times$ 9.6	$E^pE^pii \times EE^pI^di$	29	..	4	10	6	9
433 $\times$9.6 & 9.10	$E^pE^pii \times EE^pI^di$	15	1	6	4	2	2
	Total	44	1	10	14	8	11
Black-spotted $\times$ F_1 blue roan							
433 $\times$ 8.7 & 8.8	$E^pE^pii \times EEI^di$	23	—	8	15	..	..
Black-spotted $\times$ F_1 white							
433 $\times$ 103.8 & 103.9	$E^pE^pii \times EE^pIi$	27	19	..	2		6

*F_2 boar 141 came from the mating of boar 8.5 with sow 9.6. Boar 433 was an F_3 son of boar 141.

red roan, and black-spotted. By this same boar, sow 103.8 produced all expected phenotypes except the black-spotted class in a total of only 17 pigs, but when mated to boar 9.5 she produced only three of the five expected classes in 38 pigs, the red roan and black-spotted classes being absent. Furthermore, the observed numbers among the combined F_2 progenies (61 white, 27 blue roan, 15 black, 3 red roan, 1 black-spotted) deviate significantly from the expected numbers on the basis of a 8:3:3:1:1 ratio ($P = 0.02$). These results cumulatively suggest that boar 9.5 lacked the black-spotting gene E^p and hence was EEI^di.

This interpretation is strengthened when the progenies of boars 8.5 and 9.5 are considered separately. The offspring of boar 8.5 do not deviate significantly from expectation from the above ratio ($\chi^2 = 6.79$, d.f. $= 4$, $P = 0.16$), and the offspring of boar 9.5 conform satisfactorily to the 2:1:1 ratio expected if he was EEI^di ($\chi^2 = 1.32$, d.f. $= 2$, $P = 0.52$). Since I and I^d as well as i were involved in all the above matings, it may be concluded, therefore, that white (I), roaning (I^d), and full color (i) form a series of triple alleles, which are probably independent of the E locus. The offspring of boar 8.5 show moreover that the error in distinguishing between I and I^d was small, and few, if any, blue roans or red roans (I^di) were mistaken for whites.

The two white F_1 sows whose F_2 offspring have just been described (i.e. 103.8 and 103.9) later were mated to one of the black-spotted boars (433) who by blue roan F_1 sows 9.6 and 9.10 had produced blue roans, red roans and whites in addition to black and black-spotted pigs. By sows 103.8 and 103.9 this same boar produced 19 whites, 2 blacks and 6 black-spotted sandies, but no blue roans or red roans. This proves almost conclusively that both sows lacked the gene I^d, as expected.

TABLE III. Distribution of progenies from different types of matings involving F_2, backcross and later generation animals. Expected numbers are given in parentheses.

Matings	Genotypes	Expected	Number of pigs: White	Blue roan	Black	Red roan	Bl. sp.	X^2	P
Black-spotted × white	$E^pEP^pii \times E^pEP^pII$	All white	16 (16.0)						
	$E^pEP^pii \times E^pEP^pIi$	1:1	30 (29.5)				29 (29.5)	0.02	0.90
	$E^pEP^pii \times EEP^pIi$	2:1:1	43 (43.0)		27 (21.5)		16 (21.5)	2.81	0.25
	$E^pEP^pii \times E^pEP^pI^di$	1:1				9 (12.0)	15 (12.0)	1.50	0.22
Black-spotted × blue roan	$E^pEP^pii \times EEI^di$	1:1		14 (12.0)	10 (12.0)			0.67	0.43
	$E^pEP^pii \times EEP^pI^di$	1:1:1:1		5 (5.2)	6 (5.2)	[a] 7 (5.2)	3 (5.2)	1.67	0.65
Black-spotted × black	$E^pEP^pii \times EEii$*	All black			87 (87.0)				
	$E^pEP^pii \times EEP^pii$*	1:1			32 (32.5)		33 (32.5)	0.02	0.90
Black-spotted × black-spotted	$E^pEP^pii \times E^pEP^pii$	All black-sp.					305 (305.0)		
Black × white	$EEii \times EEP^pIi$	1:1	48 (53.0)		58 (53.0)			0.94	0.44
	$EEP^pii \times E^pEP^pIi$	2:1:1	4 (5.0)		1 (2.5)		5 (2.5)	3.60	0.17
	$EEP^pii \times EEP^pIi$	4:3:1	60 (74.5)		62 (55.9)		27 (18.6)	7.20	0.03
	$EEP^pii \times E^pEP^pI^di$	1:1:1:1		3 (4.2)	4 (4.2)	[b] 6 (4.2)	4 (4.2)	1.12	0.76
Black × blue roan	$EEii \times EEP^pI^dI^d$	All blue roan		8 (8.0)					
	$EEP^pii \times EEI^dI^d$	" " "		16 (16.0)					
	$EEii \times EEI^di$	1:1		10 (9.5)	9 (9.5)			0.05	0.82
	$EEii \times EEP^pI^di$*	1:1		19 (19.0)	19 (19.0)			0.00	1.00
	$EEP^pii \times EEI^di$	1:1		15 (13.5)	12 (13.5)			0.33	0.58
	$EEP^pii \times EEP^pI^dI^d$	3:1		3 (3.8)		2 (1.2)		0.60	0.45
	$EEP^pii \times EEP^pI^di$	1:3:3:1	2 (1.6)	6 (4.9)	1 (4.9)		4 (1.6)	6.90	0.08
Black × black	$EEii \times EEii$	All black			108 (108.0)				
	$EEP^pii \times EEii$	" "			275 (275.0)				
	$EEP^pii \times EEP^pii$	3:1			412 (404.2)		127 (134.8)	0.59	0.46
Blue roan × white	$EEP^pI^di \times E^pEP^pI^di$	3:3:1:1	[c] 5 (3.8)	3 (3.8)	1 (1.2)		1 (1.2)	0.67	0.88
Blue roan × blue roan	$EEP^pI^di \times EEP^pI^di$	9:3:3:1		1 (1.7)	1 (0.6)	— (0.6)	1 (0.2)	—	—
White × white	$EEP^pIi \times EEP^pIi$	12:3:1	26 (25.5)		5 (6.4)		3 (2.1)	0.67	0.71
	$EEP^pIi \times E^pEP^pIi$	6:1:1	7 (9.0)		3 (1.5)		2 (1.5)	2.11	0.36
White × black	$E^pEP^pII \times EEii$	All white	71 (71.0)						
	$E^pEP^pII \times EEP^pii$	" "	62 (62.0)						

*Cross made reciprocally

[a]One was white. [b]Two were white. [c]One was red roan.

Types of Progeny from Other Matings

In addition to the matings described above, a large amount of data have been obtained from matings involving F_2, backcross and later generation animals. Since only a few of the matings were made for the purpose of verifying the relations of the genes considered here, it was not possible to assign genotypes to all animals whose genotypes were not specifically determined by those of their parents or by their individual appearance. While all matings involving such animals gave results consistent with those expected on the basis of our factorial hypothesis, the procedure followed here was to use only animals which could be assigned a specific genotype with reasonable certainty. A summary of the matings involving such animals is shown in Table III.

Of the 29 types of matings listed in Table III, five showed segregation for both E–E^p and I–i, five for both E–E^p and I^d–i, three for E–E^p only, two for I–i only and five for I^d–i only. The remaining nine types all produced only one kind of pigs. Although some of the latter type matings, as well as some of those which showed segregation for only one pair of factors, may have been capable of producing more than one or two kinds of pigs, the results of the various matings indicate rather conclusively that few if any of the animals represented therein were misclassified with respect to the genetic constitutions assigned. As a matter of fact there is only one case (*i.e.* $EE^pii \times EE^pIi$) where the deviations border on significance ($P = 0.03$), there being an excess of black and black-spotted and a deficiency of white. Since all the other matings gave deviations within a range attributable to random errors, the poor fit in this case may be attributed to extreme chance deviations.

Summary

Data on the inheritance of the color genes differentiating the Landrace from the Hampshire are presented. All Hampshires used in the crosses lacked the white belt which is characteristic of that breed, being entirely black. The Landrace animals, all of which came from the same source as those used in other crosses described previously, were white or white with a few skin spots.

A previously unreported gene was found in three of the nine Landrace animals used in the original crosses. This gene, to which the symbol I^d (dilution) has been given, behaves as an intermediate allele of I (white) and its allele i (intense color) in animals possessing the gene for self black (E). In such animals, black is modified to a blue roaning pattern similar to that of the Sapphire hog.

When combined with the black spotting gene E^p, I^d was found to suppress the production of black in the same manner as its allele I. In contrast to I the gene I^d, on the other hand, does not inhibit completely the production of red color. It appears to reduce red to a red roaning pattern, which is similar to the blue roaning pattern except for the presence of red instead of black hairs.

As the black color of the Hampshire behaves as a simple dominant to the black spotting gene E^p carried hypostatically by the Landrace, the conclusion is that the black of the Hampshire probably is due to the same fundamental gene for black (E) as that previously demonstrated in the Large Black. The evidence also indicates that the Hampshire and Large Black have the same or similar red intensity factors, lacking in whole or in part in the Landrace.

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